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**Appendix B**

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**PRESS RELEASE  
FOR IMMEDIATE RELEASE**

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**HUMAN GENOME SCIENCES INITIATES TRIAL OF A NEW DRUG FOR  
SYSTEMIC LUPUS ERYTHEMATOSUS AND OTHER AUTOIMMUNE  
DISEASES**

-- Human Antibody Drug, LymphoStat-B™, May Suppress Overactive Immune System --

ROCKVILLE, Maryland -- November 1, 2001 -- Human Genome Sciences, Inc. (Nasdaq: HGSI) announced today that the U.S. Food & Drug Administration has approved its Investigational New Drug application to begin clinical trials of LymphoStat-B™, as a potential new treatment for autoimmune diseases. Human Genome Sciences will now proceed with a Phase 1 clinical trial in patients with systemic lupus erythematosus. The trial will be a multi-center, dose-escalation study to determine the safety and pharmacology of the drug in adult patients who are receiving standard therapies. In the future, LymphoStat-B may also be tested in patients with other autoimmune diseases, such as rheumatoid arthritis, immune thrombocytopenic purpura, and Sjogren's syndrome.

Systemic lupus erythematosus is a serious, life-threatening disease. Between 200,000 and 500,000 people are diagnosed with systemic lupus each year in the United States alone. The disease affects between eight and ten times as many women as men. It may occur at any age, but appears mostly in young people between the ages of fifteen and forty-five. Symptoms may include extreme fatigue, painful and swollen joints, unexplained fever and kidney problems. Systemic lupus erythematosus can lead to arthritis, kidney failure, heart and lung inflammation, central nervous system abnormalities, inflammation of the blood vessels and blood disorders.

LymphoStat-B acts by inactivating a natural immune stimulator, the B Lymphocyte Stimulator (BLyS™). Many patients that suffer from systemic lupus erythematosus and rheumatoid arthritis have elevated levels of BLyS in their blood or joint fluid (see "High Levels of BLyS Implicated In Lupus And Rheumatoid Arthritis Patients," October 30, 2000, at [www.hgsi.com/news/press/00-10-30\\_BLyS\\_ACR.html](http://www.hgsi.com/news/press/00-10-30_BLyS_ACR.html)).<sup>1</sup> Laboratory studies show that LymphoStat-B can reverse the immune stimulatory effects of BLyS.

William Stohl, M.D., Ph.D., Professor of Medicine, Division of Rheumatology, University of Southern California, said, "Studies in laboratory models of lupus, as well as in human lupus patients, suggest that

elevated levels of BLyS may have a significant role in systemic lupus erythematosus. Furthermore, laboratory studies of rheumatoid arthritis and studies in human rheumatoid arthritis patients raise the possibility that elevated BLyS levels may contribute to this disease as well. LymphoStat-B, by virtue of its ability to neutralize BLyS activity, may prove to be an effective treatment approach for patients suffering from systemic lupus, rheumatoid arthritis and possibly other autoimmune diseases."

Robert P. Kimberly, M.D., Professor of Medicine and Microbiology, and Director, Division of Clinical Immunology and Rheumatology, The University of Alabama at Birmingham, said, "Autoimmune diseases represent the third-greatest clinical burden after cardiovascular disease and cancer, and currently have no cure. These diseases are complex and often devastating chronic illnesses. Patients with systemic lupus, in particular, can experience any number of symptoms that can flare unexpectedly on average two to three times a year. For patients with severe symptoms, typically involving their internal organs, this disease can sometimes be fatal. There is great need for further research into the causes of these diseases, as well as for new treatment options that can help control disease progression and improve patients' quality of life."

*BAFF*  
LymphoStat-B is a fully human monoclonal antibody that binds to and inactivates the BLyS protein. The drug was made discovered in a collaboration between scientific teams at Human Genome Sciences and Cambridge Antibody Technology. The drug for clinical trials will be made in Human Genome Sciences' newly completed clinical trial manufacturing facility, located in Rockville, Maryland. Human Genome Sciences holds commercial rights to the drug.

Craig A. Rosen, Ph.D., Executive Vice President, Research and Development, Human Genome Sciences, Inc., said, "LymphoStat-B represents a significant advance for patients with systemic lupus erythematosus. It LymphoStat-B represents several firsts. The drug provides the first hope in many years for patients with systemic lupus erythematosus. It is also also our first compound to enter human clinical trials that is a monoclonal antibody. We now have now demonstrated our the capacity to discover, optimize, manufacture and develop human monoclonal antibody drugs."

He continued, "To my knowledge, LymphoStat-B is also the first genomics-based antibody drug to enter clinical trials. LymphoStat-B binds to and inactivates the BLyS protein. BLyS was discovered by Human Genome Sciences using genomic methods. BLyS is currently being developed on its own as a drug to treat certain immune deficiency diseases."<sup>2</sup> (See "Human Genome Sciences To Initiate Human Clinical Trials of BLyS," June 23, 2000, at [www.hgsi.com/news/press/00-06-23\\_BLySIND.html](http://www.hgsi.com/news/press/00-06-23_BLySIND.html); and "Human Genome Sciences Announces Trial For Treatment of Immunoglobulin-A Deficiency," September 19, 2001 at [www.hgsi.com/news/press/01-09-19\\_BLyS\\_IgA.html](http://www.hgsi.com/news/press/01-09-19_BLyS_IgA.html))

David C. Stump, M.D., Senior Vice President, Drug Development, Human Genome Sciences, Inc., said, "LymphoStat-B has the potential to treat a family of serious autoimmune diseases, including systemic lupus erythematosus and rheumatoid arthritis. If the initial Phase 1 trials of the drug are successful, we hope to be able to add new indications for additional trials for patients with rheumatoid arthritis and other autoimmune diseases, such as immune thrombocytopenic purpura, and Sjogren's syndrome. These studies should also shed much light on the role of hyperactive B-cell immunity, and of BLyS in particular, in the origin of a broad family of autoimmune diseases."

William A. Haseltine, Ph.D., Chairman and Chief Executive Officer, Human Genome Sciences, Inc., said, "I am delighted that we can now begin trials of LymphoStat-B for the treatment of systemic lupus erythematosus and other autoimmune diseases. As a family, these diseases cause immense suffering to millions of patients worldwide. LymphoStat-B provides a new, fresh, rational, mechanism-based approach for the treatment of these diseases."

He added, "LymphoStat-B also represents a significant milestone for our company. It is the sixth compound that we have entered into clinical trials. It is also our first antibody-based drug. As such, it demonstrates the powerful synergy achieved by combining genomic and antibody technology. I believe that this first genomics-based antibody drug represents a new era in medicine."

For additional information on Human Genome Sciences, please visit our web site at [www.hgsi.com](http://www.hgsi.com). For more information on LymphoStat-B, see [www.hgsi.com/products/LSB.html](http://www.hgsi.com/products/LSB.html). For more information on BLyS (B lymphocyte stimulator), see [www.hgsi.com/products/BLyS.html](http://www.hgsi.com/products/BLyS.html). For more information on autoimmune disorders, see [www.hgsi.com/news/press/background\\_ad.html](http://www.hgsi.com/news/press/background_ad.html). For more information on systemic lupus erythematosus, see [www.hgsi.com/news/press/background\\_lupus.html](http://www.hgsi.com/news/press/background_lupus.html). Copies of HGS press releases are also available by fax 24 hours a day at no charge by calling 800/758-5804, ext. 121115.

For additional information on autoimmune disorders, lupus or rheumatoid arthritis, also visit the National Institute of Arthritis and Musculoskeletal and Skin Diseases at [www.niams.nih.gov](http://www.niams.nih.gov), The Lupus Foundation of America at [www.lupus.org](http://www.lupus.org), or the Arthritis Foundation at [www.arthritis.org](http://www.arthritis.org).

Health professionals or patients interested in inquiring about the LymphoStat-B trial or any other study involving HGS products are encouraged to inquire via the Contact Us section of the Human Genome Sciences web site, [www.hgsi.com](http://www.hgsi.com), or by calling us at (301) 610-5790, extension 3550.

Human Genome Sciences is a company with the mission to treat and cure disease by bringing new gene-based drugs to patients.

HGS, Human Genome Sciences, LymphoStat-B and BLyS are registered trademarks of Human Genome Sciences, Inc.

This announcement contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. The forward-looking statements are based on Human Genome Sciences' current intent, belief and expectations. These statements are not guarantees of future performance and are subject to certain risks and uncertainties that are difficult to predict. Actual results may differ materially from these forward-looking statements because of the company's unproven business model, dependence on new technologies, uncertainty and timing of clinical trials, ability to develop and commercialize products, dependence on collaborators for services and revenue, substantial indebtedness, intense competition, uncertainty of patent and intellectual property protection, dependence on key management, uncertainty of regulation of products, dependence on key suppliers, the impact of future alliances or transactions and other risks that may be described in the company's filings with the Securities and Exchange Commission. Existing and prospective investors are cautioned not to place undue reliance on these forward-looking statements, which speak only as of today's date. Human Genome Sciences undertakes no obligation to update or revise the information contained in this announcement whether as a result of new information, future events or circumstances or otherwise.

Footnotes:

1. See "High Levels of BLyS Implicated In Lupus And Rheumatoid Arthritis Patients" at [www.hgsi.com/news/press/00-10-30\\_BLyS\\_ACR.html](http://www.hgsi.com/news/press/00-10-30_BLyS_ACR.html).
2. See "Human Genome Sciences To Initiate Human Clinical Trials of BLyS" at [www.hgsi.com/news/press/00-06-23\\_BLySIND.html](http://www.hgsi.com/news/press/00-06-23_BLySIND.html), and "Human Genome Sciences Announces Trial For Treatment of Immunoglobulin-A Deficiency" at [www.hgsi.com/news/press/01-09-](http://www.hgsi.com/news/press/01-09-)

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# HUMAN GENOME SCIENCES

## PRODUCTS

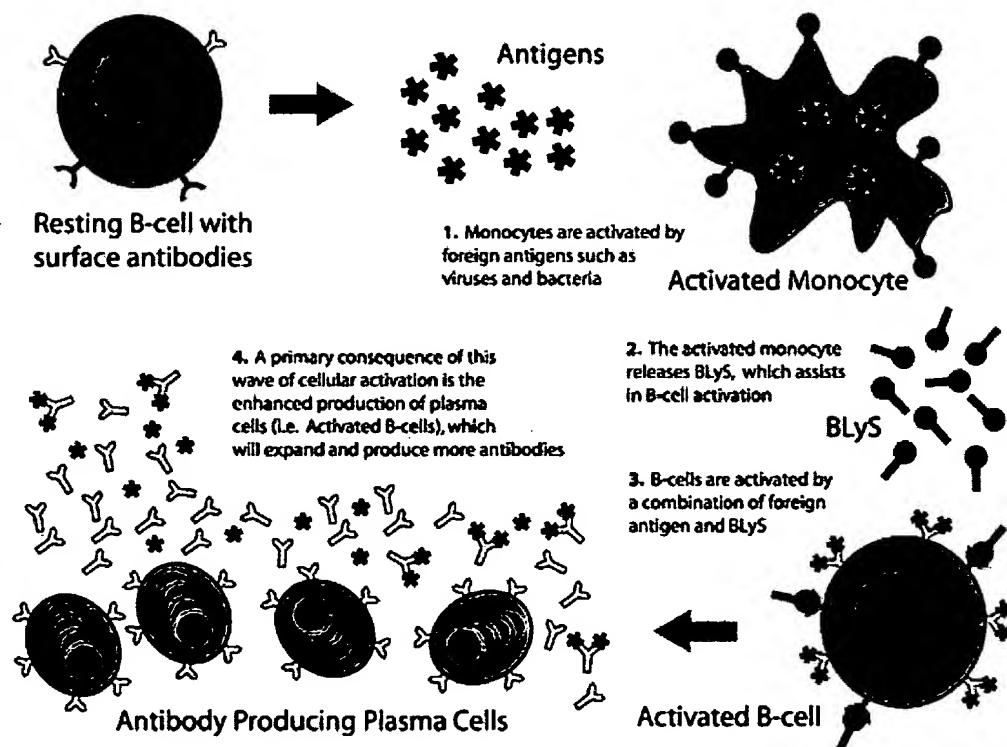


Giambattista Tiepolo  
*Mercury, Messenger of the Gods*, 1653

## B Lymphocyte Stimulator (BLyS)

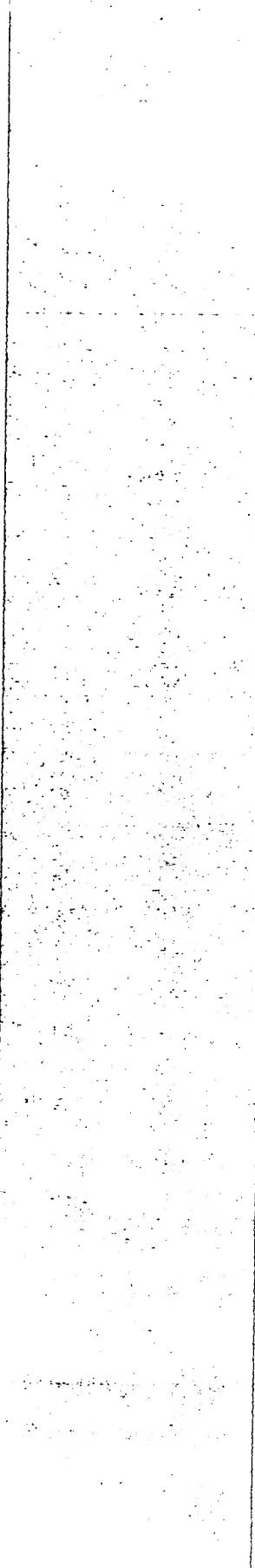
### Background

In July 1999, Human Genome Sciences (HGS) reported the discovery of a novel human protein called B Lymphocyte Stimulator, or BLyS. (1) BLyS stimulates immune system cells called B cells to mature into plasma B cells, which produce antibodies (see Fig. 1). Plasma B cells and the antibodies they produce constitute a critical part of the body's defense against infections and cancer.



**Fig. 1: BLyS released from monocytes activates resting B cells, stimulating them to become plasma B cells that secrete antibodies. (See [Detailed Image](#))**

The discovery of BLyS may lead to therapies for several diseases that involve B cells, including immune deficiencies, autoimmune disease and B cell tumors. Human Genome Sciences' drug development teams are advancing several therapeutic concepts based on the discovery of BLyS: BLyS therapeutic protein, human monoclonal antibodies targeting BLyS, and



radiolabeled BLyS.

### **How BLyS Works**

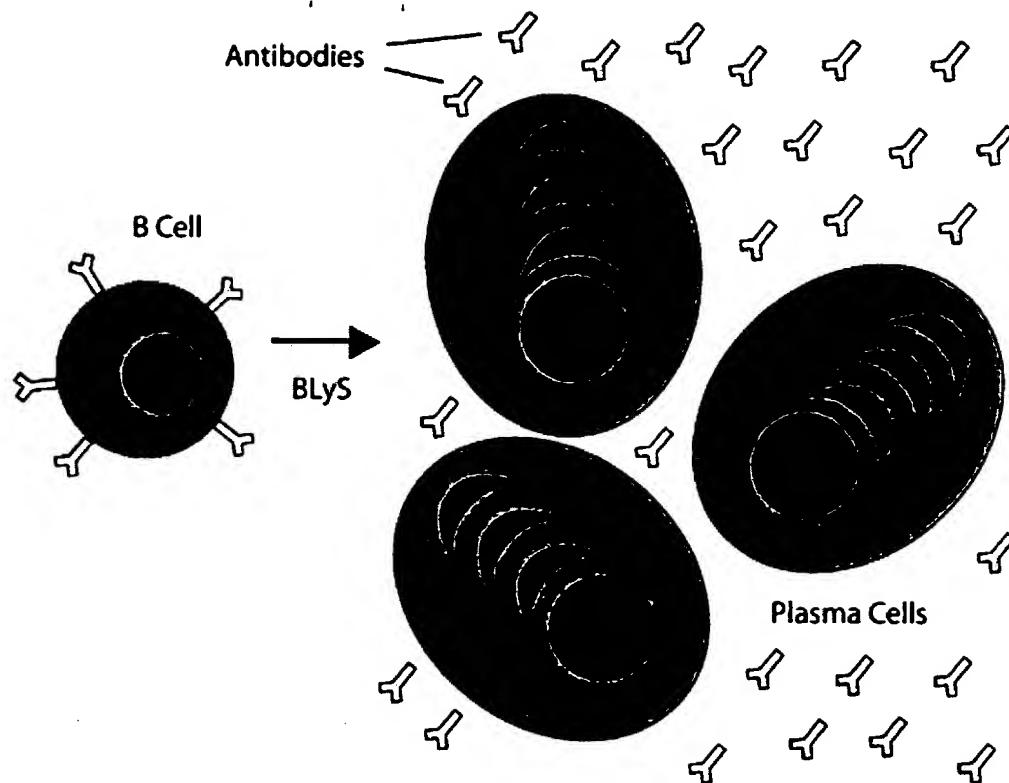
BLyS is made by immune-cells called monocytes and macrophages. When monocytes and macrophages are activated, BLyS is released and binds to a receptor found only on B cells. B cells arise from stem cells that do not themselves produce antibodies. When BLyS binds to its receptor on B cells, they mature into antibody-secreting plasma B cells. As a result, the number of antibodies in the patient's plasma increases.

When antibodies recognize foreign molecules, immune-cells target the molecules for destruction. Without plasma B cells and antibodies, the body is largely unprotected against pathogens, and infectious disease may follow.

### **TURNING BLyS INTO TECHNOLOGY FOR FIGHTING HUMAN DISEASE**

#### **BLyS Therapeutic Protein for Immunodeficiency**

Immunodeficiency disorders are a diverse group of conditions caused by one or more immune system defects. They are characterized by increased susceptibility to infections with consequent severe, acute, recurrent or chronic disease. Physicians recognize more than 70 different primary immunodeficiencies—ones that are caused by abnormalities in the development of immune system cells. The Human Genome Sciences BLyS Therapeutic Protein Program is developing BLyS protein as a therapeutic agent that may be valuable in the treatment of these immunodeficiencies (see Fig. 2).



**Fig. 2: BLyS protein stimulates B cells to produce antibodies.**

#### **Primary Immunodeficiency: CVID**

Common variable immunodeficiency (CVID) is a group of immunodeficiency syndromes in which B cell immunity is abnormal. Most patients have normal or near-normal numbers of circulating B cells, but the cells fail to differentiate into

effective plasma B cells. As a result, patients have low or undetectable amounts of serum antibodies. The condition may result from insufficient stimulation of B cells rather than from a failure intrinsic to B cells. (2)

There are several thousand CVID patients in the United States and Europe. CVID occurs equally in both genders. Most patients experience acute, recurring bacterial infections, including pneumonia, bronchitis and sinusitis. (3) Current treatment involves regular administration of intravenous antibodies, which are prepared from pooled blood samples from thousands of individual donors.

BLyS protein may boost antibody levels in patients with CVID, as well as in other immunodeficiency conditions that effectively mimic CVID. Human Genome Sciences scientists have found in laboratory studies that BLyS boosts antibody production in B cells isolated from some CVID patients.

Human Genome Sciences recently completed enrollment in a Phase 1 trial evaluating the safety and pharmacology of BLyS in patients with CVID.

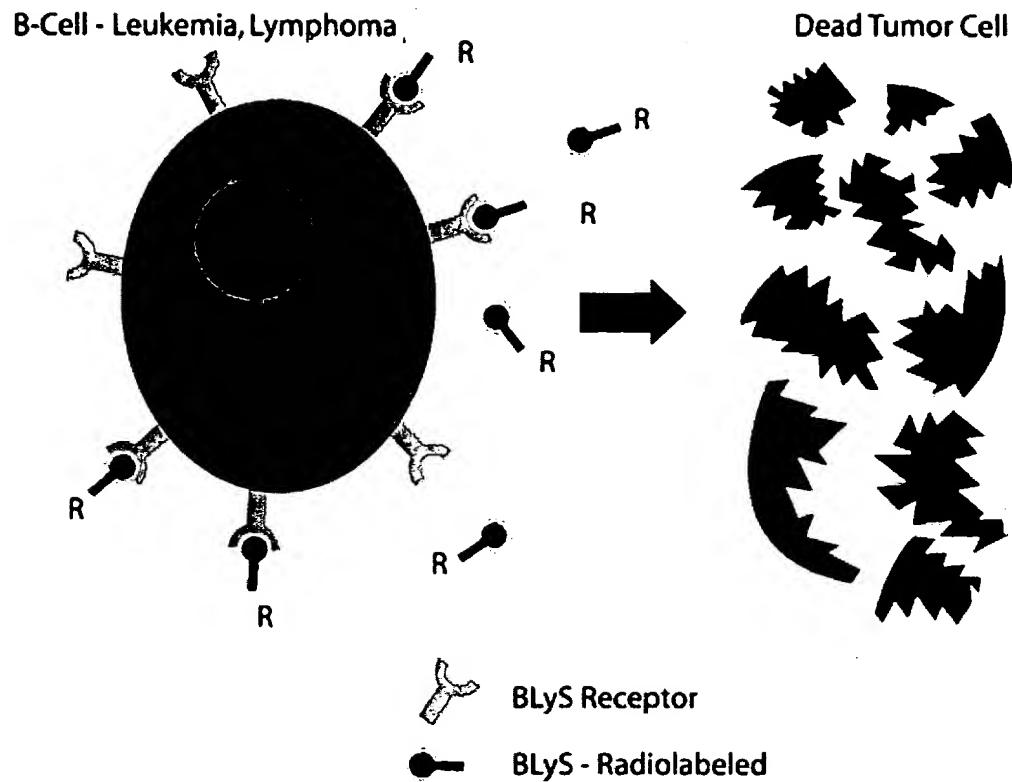
### **Immunoglobulin-A Deficiency**

Immunoglobulin-A deficiency is a disorder of the immune system characterized by increased susceptibility to infection. Patients with this disease fail to produce normal amounts of one particular type of antibody, called immunoglobulin-A. This type of antibody provides the first line of defense for the inner surfaces of the body against infections of the lung, the intestine, the mouth, the urogenital tract and other areas lined by mucosal membranes. It is believed that immunoglobulin-A deficiency may result from the failure of one type of immune cell called the B lymphocyte to mature into plasma cells that produce immunoglobulin-A antibodies.

Immunoglobulin-A deficiency is the most common disorder of the antibody system. Not all patients are aware that they have this immune deficiency, which accounts for the wide variations in estimates of the number of affected people. Symptomatic patients suffer from recurrent and serious infections, including infections of the gastrointestinal tract, lungs and sinuses, as well as allergic disorders, epilepsy and cancer.

There are currently no available therapies that address the underlying cause of immunoglobulin-A deficiency. Treatment with BLyS may help immunoglobulin-A deficient patients produce their own antibodies. The BLyS protein is known to be able to stimulate B cells to produce immunoglobulin-A antibodies as well as other types of antibodies. Preclinical studies have also shown that BLyS proteins can stimulate the B cells of some immunoglobulin-A deficient patients to produce larger quantities of immunoglobulin-A antibodies than normal.

Human Genome Sciences is conducting a Phase 1 trial to evaluate the safety and pharmacology of BLyS in patients with IgA deficiency.



**Fig. 3: BlyS protein attached to a radioisotope such as iodine-131 may bind to BlyS receptors on cancerous B cells and kill the cells with low doses of radiation.**

#### **Radiolabeled BlyS for B-cell malignancies**

BlyS linked to radionuclides have a potential application as therapy for B-cell malignancies (see Fig. 3). Such malignancies are responsive to radiation, and radiotherapy is an important part of the treatment plan for many patients with these diseases. A drug consisting of BlyS linked to a source of radiation would bind only to B cells, so low doses of radiation would be effective at killing such cells (see Fig. 4). Human Genome Sciences is working to evaluate technologies that will allow a radioisotope to be linked to BlyS to create drugs that bind to and kill B-cells.

#### **B Cell Cancers**

B cells are centrally involved in certain types of cancer, including non-Hodgkin's lymphoma, chronic lymphocytic leukemia and multiple myeloma. In these diseases, B cells become malignant and grow in an unregulated fashion.

Non-Hodgkin's lymphoma is the fifth-most-common type of cancer diagnosed in the U.S. each year. Chronic lymphocytic leukemia is the most common form of leukemia. Multiple myeloma is a deadly form of B cell cancer with a five-year survival rate of 28 per cent. New therapies are needed to improve

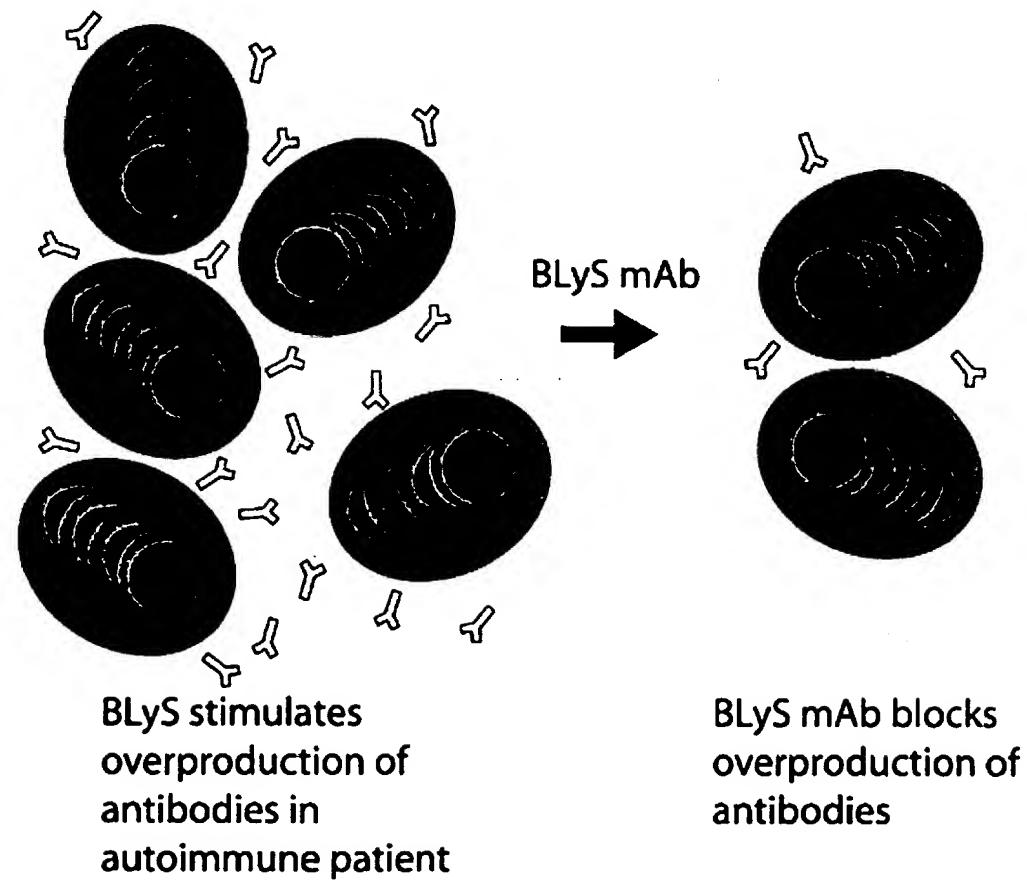
survival rates and bring cures to more patients with these forms of cancer.

Human Genome Sciences is conducting a Phase 1 trial to evaluate the safety and pharmacology of LymphoRad<sup>TM 131</sup>, a radioiodinated form of BLyS, in patients with multiple myeloma. For more information, see [LymphoRad](#).

### BLyS Antagonists for Autoimmune Disease

#### Autoimmune Disease

The immune system has to distinguish the body's own cells and tissues from those of pathogens so that it can avoid attacking itself while maintaining a diverse repertoire of antibodies. Abnormalities in the induction or maintenance of self-tolerance—the process that prevents the immune system from attacking the body's own tissues—can lead to inflammatory immune responses developing against self-antigens and thus to autoimmune disease. B cells that produce antibodies that recognize parts of the normal body play an important role in many autoimmune diseases including systemic lupus erythematosus and rheumatoid arthritis.



**Fig. 4: Anti-BLyS molecules prevent BLyS from having its normal effect**

and so block the overproduction of antibodies.

Human Genome Sciences scientists are creating human monoclonal antibodies that bind to BLyS and inactivate the BLyS protein. It is already known that overproduction of BLyS in animals leads to a lupus-like disease. (4) Experiments in models of autoimmune disease suggest that such BLyS antagonists may reduce the body's ability to produce harmful self-reactive antibodies, with consequent benefits for patients.

Human Genome Sciences is conducting a Phase 1 trial to evaluate the safety and pharmacology of LymphoStat-B<sup>TM</sup>, a human monoclonal antibody to BLyS, in patients with systemic lupus erythematosus. For more information, see [LymphoStat-B](#).

### How BLyS Was Discovered

#### A Functional Proteomics Success Story

Human Genome Sciences scientists discovered BLyS (B Lymphocyte Stimulator) via functional proteomics, the study of the natural function and medical use of proteins discovered by genomic technology.

For decades, scientists sought a biological signal that stimulates immune-cells called B cells to become plasma B cells, which produce antibodies. Because biological signals are often secreted proteins, Human Genome Sciences scientists were studying a group of about 400 human proteins in the Human Genome Sciences database whose DNA sequences suggested that they were secreted. Each protein was purified and tested for the ability to stimulate B cell growth. One protein, BLyS, had a powerful effect on B cells.

Just as important, BLyS lacked effects on other cells. In pre-clinical experiments, BLyS was found to increase the production of antibodies, immediately suggesting medical applications in the treatment of immunodeficiencies and possibly other conditions. The Human Genome Sciences scientists published the BLyS research in *Science* magazine in July 1999. (1)

The Human Genome Sciences functional proteomics program currently examines the biological activity of 9,000 human secreted proteins in the search for those that may serve as useful drugs. Human proteins identified through genomics may have medical properties superior to conventional small-molecule drugs, and may enter clinical trials more quickly, because less medicinal chemistry research is needed to turn them into product candidates. Human antibodies to human genes and proteins may prove to be important medicines, and Human Genome Sciences is pursuing medicines based on antibodies to BLyS through several collaborations.

### Conclusions

The discovery of BLyS, a long-sought key immune system regulator with

multiple possible uses in medicine, exemplifies the power of genomics to find molecules with therapeutic potential. Standard biochemical techniques had failed to identify BLyS, but systematic screening of proteins produced by candidate human genes revealed its identity and its powerful influence on the immune system. Genomics and proteomics may yield many additional potent molecules with medical applications.

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1. Paul A. Moore, Ornella Belvedere, Amy Orr, Krystyna Pieri, David W. LaFleur, Ping Feng, Daniel Soppet, Meghan Charters, Reiner Gentz, David Parmelee, Yuling Li, Olga Galperina, Judith Giri, Viktor Roschke, Bernardetta Nardelli, Jeffrey Carrell, Svetlana Sosnoveva, Wilbert Greenfield, Steven M. Ruben, Henrik S. Olsen, James Fikes, and David M. Hilbert. BLyS: Member of the Tumor Necrosis Factor Family and B Lymphocyte Stimulator. *Science*, Vol. 285, Number 5425, 260-263. July 9, 1999.
2. F. Rosen et. al. The Primary Immunodeficiencies, A Review Article. *New England Journal of Medicine* 333: 7, August 17, 1995.
3. Immune Deficiency and Allied Disorders: Clinical Updates, Immune Deficiency Foundation Vol. II, Issue 1, July 1995.
4. Mice transgenic for BAFF develop lymphocytic disorders along with autoimmune manifestations. Mackay F, Woodcock SA, Lawton P, Ambrose C, Baetscher M, Schneider P, Tschopp J, and Browning JL. *J Exp Med* 1999 Dec 6; 190(11): 1697-1710.

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Several peer-reviewed papers on BLyS have been published by Human Genome Sciences scientists. To read an abstract of the papers, click on the titles below:

[BLyS: member of the tumor necrosis factor family and B lymphocyte stimulator](#)

[Click for additional references](#)

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To view announcements on BLyS, click on the following:

[Human Genome Sciences Reports Progress in Clinical Trials of Five Drugs at JP Morgan H&Q Conference - January 6, 2003](#)

[Human Genome Sciences Announces Clearance of Investigational New Drug Application for LymphoRad<sup>131</sup>, A New Anticancer Drug for the Treatment of B-Cell Tumors, May 14, 2002](#)

Human Genome Sciences Files Investigational New Drug Application for LymphoRad<sup>131</sup>, January 23, 2002

Human Genome Sciences Initiates Trials of a New Drug for Systemic Lupus Erythematosus and Other Autoimmune Diseases — November 1, 2001

Human Genome Sciences Announces Trial For Treatment of Immunoglobulin-A Deficiency - September 19, 2001

Human Genome Sciences Receives Orphan Drug Designation for BLyS Therapeutic Protein for Treatment of Common Variable Immunodeficiency - February 27, 2001

High Levels of BLyS Implicated in Lupus and Rheumatoid Arthritis Patients - October 30, 2000

Human Genome Sciences and Cambridge Antibody Technology Commit to Exclusive Development of Anti-BLyS Antibodies - October 30, 2000

Human Genome Sciences and Dow Agree To Develop HGS' Radiolabeled B-Lymphocyte Stimulator - October 30, 2000

Human Genome Sciences to Initiate Human Clinical Trials of BLyS - June 23, 2000

Human Genome Sciences Announces the Discovery of a Novel Immune Stimulant - July 8, 1999

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Background information for BLyS is available for the following indications:

Common Variable Immunodeficiency

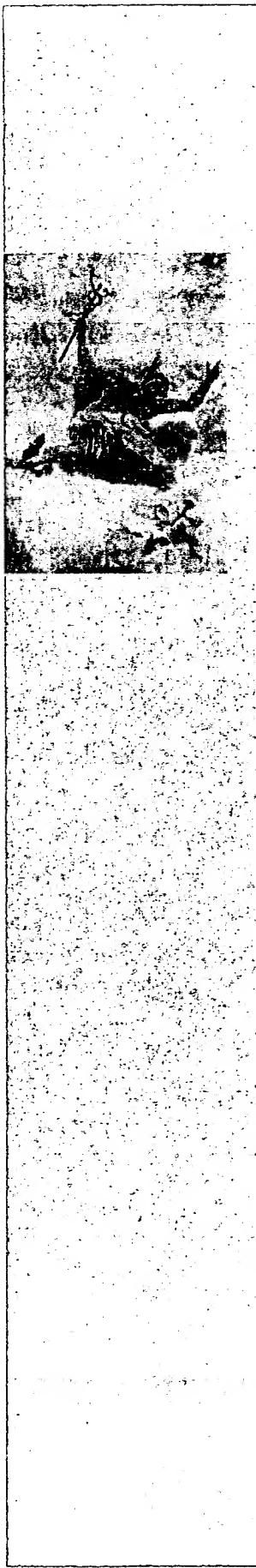
Immunoglobulin-A Deficiency

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## HUMAN GENOME SCIENCES

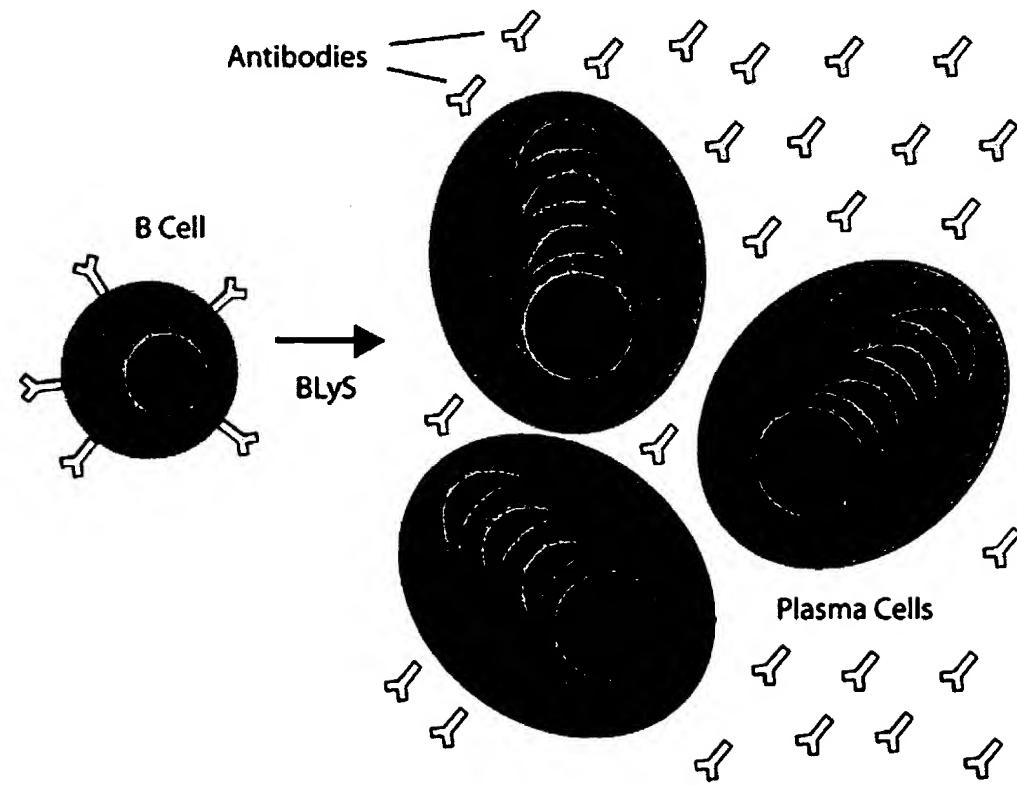
### PRODUCTS



#### LymphoStat-B (Human Monoclonal Antibody To B Lymphocyte Stimulator / BLyS)

##### Background

LymphoStat-B™ is a fully human, monoclonal antibody designed to inhibit the biological activity of B Lymphocyte Stimulator, or BLyS™. BLyS is a naturally occurring protein that stimulates B lymphocyte cells to develop into mature plasma B cells, which produce antibodies<sup>1</sup>, the body's first line of defense against infection (see Fig. 1). Laboratory studies have indicated that higher than normal levels of BLyS may trigger autoimmune diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis.<sup>2, 3, 4, 5</sup> Autoimmune diseases are diseases in which the body is attacked by its own immune system.





### **Fig. 1: B Lymphocyte Stimulator (BLyS) protein stimulates B cells to produce antibodies**

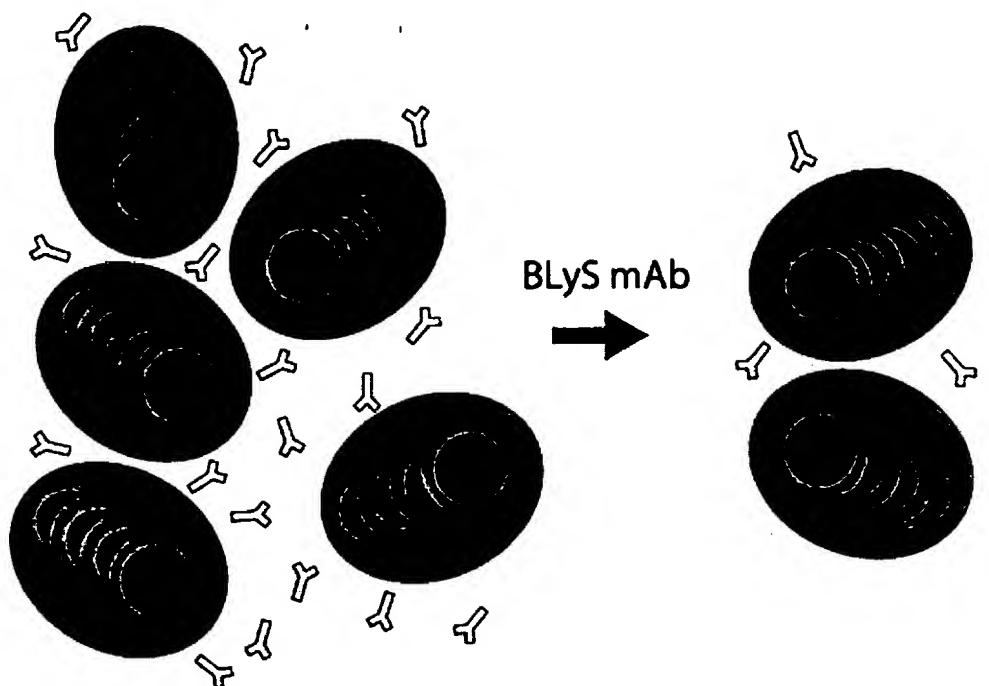
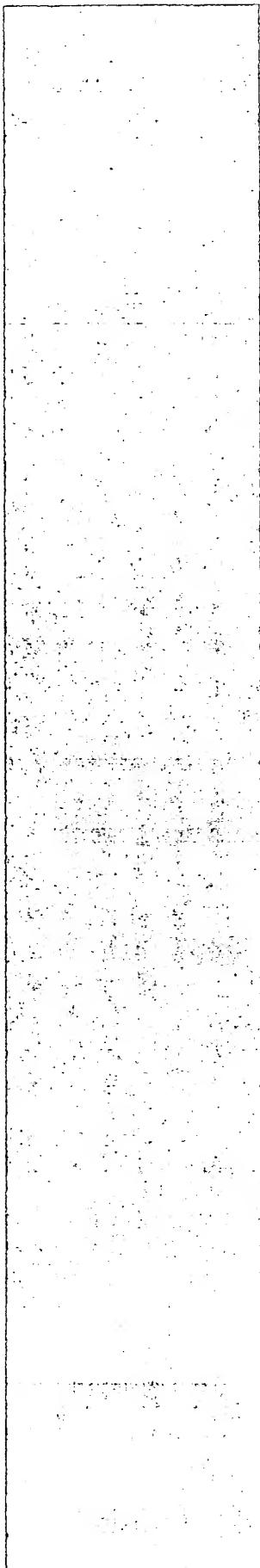
Human Genome Sciences is conducting a Phase 1 clinical trial that will provide safety and pharmacologic data for LymphoStat-B as a potential treatment for patients with systemic lupus erythematosus (SLE). Systemic lupus is a chronic, life-threatening autoimmune disease in which the immune system mounts an attack on the body's own cells and tissues.

Human Genome Sciences announced the discovery of BLyS and provided a preliminary description of its activity in July 1999.<sup>1</sup> Following the discovery of BLyS, Human Genome Sciences initiated a program to identify antibodies that would inhibit BLyS, for development into potential treatments. Human Genome Sciences, in collaboration with Cambridge Antibody Technology (CAT), discovered and optimized the antibody to BLyS, LymphoStat-B, as a drug.

#### **How LymphoStat-B Works**

LymphoStat-B works by binding to BLyS and inhibiting its stimulation of B cell development and proliferation (see Fig. 2). In many autoimmune diseases, elevated levels of BLyS are believed to contribute to the production of autoantibodies—antibodies that attack and destroy the body's own healthy tissues.

Retrospective studies have shown elevated BLyS levels in the blood of patients with systemic lupus erythematosus, and in the blood and joint fluid of patients with rheumatoid arthritis.<sup>6, 7</sup> Systemic lupus erythematosus is marked by excess antibody production and abnormal B lymphocyte cell function. LymphoStat-B may be able to reduce circulating BLyS levels and autoantibody levels in patients with systemic lupus.



BLyS stimulates overproduction of antibodies in autoimmune patient

BLyS mAb blocks overproduction of antibodies

**Fig. 2: LymphoStat-B (BLyS mAb) prevents BLyS from having its normal effect and so blocks the overproduction of antibodies.**

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1. Paul A. Moore, Ornella Belvedere, Amy Orr, Krystyna Pieri, David W. LaFleur, Ping Feng, Daniel Soppet, Meghan Charters, Reiner Gentz, David Parmelee, Yuling Li, Olga Galperina, Judith Giri, Viktor Roschke, Bernardetta Nardelli, Jeffrey Carrell, Svetlana Sosnoveva, Wilbert Greenfield, Steven M. Ruben, Henrik S. Olsen, James Fikes, and David M. Hilbert. BLyS: Member of the Tumor Necrosis Factor Family and B Lymphocyte Stimulator. *Science*, Vol. 285, Number 5425, 260-263. July 9, 1999.
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2. TACI is a TRAF-interacting receptor for TALL-1, a tumor necrosis factor family member involved in B cell regulation. Xia XZ, Treanor J, Senaldi G, et al. *J. Exp. Med.* 2000; 192: 137-143.

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PCL XL error

Subsystem: KERNEL

Error: IllegalAttribute

Operator: EchoComment

Position: 24164